AMENDMENTS TO THE SPECIFICATION

Please replace the two paragraphs beginning on page 18, line 27 through page 19, line 8 with the following marked up version:

MN/CA IX was first identified in HeLa cells, derived from human carcinoma of cervix uteri, as both a plasma membrane and nuclear protein with an apparent molecular weight of 58 and 54 kilodaltons ~~(kDA)~~ **(kDa)** as estimated by Western blotting. It is N-glycosylated with a single 3kDa carbohydrate chain and under non-reducing conditions forms S-S-linked oligomers [Pastorekova et al., Virology, 187: 620-626 (1992); Pastorek et al., Oncogene, 9: 2788-2888 (1994)]. MN/CA IX is a transmembrane protein located at the cell surface, although in some cases it has been detected in the nucleus [Zavada et al., Int. J. Cancer, 54: 268-274 (1993); Pastorekova et al., supra].

A1

MN is manifested in HeLa cells by a twin protein, p54/58N. Immunoblots using a monoclonal antibody reactive with p54/58N (MAb M75) revealed two bands at 54 ~~kd~~ **kDa** and 58 ~~kd~~ **kDa**. Those two bands may correspond to one type of protein that most probably differs by post-translational processing. Herein, the phrase "twin protein" indicates p54/58N.

Please replace the paragraph on page 52, lines 29-32 with the following marked-up version:

Preferably, the intracellularly produced MN-specific antibodies are single-chain antibodies, specifically single-chain

A2

variable region fragments or ~~sFv~~ **scFv**, in which the heavy- and light-chain variable domains are synthesized as a single polypeptide and are separated by a flexible linker peptide, preferably $(Gly_4\text{-}Ser)_3$ [SEQ ID NO: 116].

Please replace the paragraph on page 58, lines 3-13 with the following marked-up version:

After subcloning, the cells isolated from transformed colonies segregated revertants. The reversion was a gradual, step-wise process; there were colonies with different degrees of reversion. After 2 passages, all the cell population became ~~a~~ morphologically indistinguishable from normal CGL1. This was due to the reversion of some cells and to the selective advantage of the revertants, which grew faster than the transformed cells. Despite repeated attempts, not even one single stably transformed cell clone was obtained. No transformed colonies were found in CGL1 cells transfected with an "empty" pMAM control plasmid. Growth of the CGL1 + pMAM.MN revertants in media supplied with 5 Fg/ml of dexamethasone for 7 days enhanced the production of MN protein, but the morphology of the cells did not return to transformed.